PB88-137765

Health and Environmental Effects Profile for 1,2-Diethylhydrazine

(U.S.) Environmental Protection Agency
Cincinnati, OH

Jan 84

U.S. Department of Commerce
National Technical Information Service
NTIS

PB86-137765

EPA/600/X-84/132
January 1984

HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR 1,2-DIETHYLHYDRAZINE

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REPRODUCED BY
U.S. DEPARTMENT OF COMMERCE
NATIONAL TECHNICAL
INFORMATION SERVICE
SPRINGFIELD, VA. 22161

TECHNICAL REPORT DATA (Please read Instructions on the reverse before completing)		
1. REPORT NO. EPA-600/X-84-132	2.	3. RECIPIENT'S ACCESSION NO. PB8 137765/AS
4. TITLE AND SUBTITLE Health and Environmental Effects Profile for 1,2-Diethylhydrazine		5. REPORT DATE January 1984
		6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)		8. PERFORMING ORGANIZATION REPORT NO
9. PERFORMING ORGANIZATION NAME AND ADDRESS		10. PROGRAM ELEMENT NO.
		11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS Environmental Criteria and Assessment Office Office of Research and Development U.S. Environmental Protection Agency Cincinnati, OH 45268		13. TYPE OF REPORT AND PERIOD COVERED
		14. SPONSORING AGENCY CODE EPA/600/22
15. SUPPLEMENTARY NOTES		

16. ABSTRACT

The Health and Environmental Effects Profile for 1,2-diethylhydrazine was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine an Acceptable Daily Intake (ADI) or a carcinogenic potency factor for 1,2-diethylhydrazine.

17. KEY WORDS AND DOCUMENT ANALYSIS		
a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
18. DISTRIBUTION STATEMENT Public	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 27
	20. SECURITY CLASS (This page) Unclassified	22. PRICE PC A02/MF A01

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

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1. INTRODUCTION

1. STRUCTURE AND CAS NUMBER

Synonyms for 1,2-diethylhydrazine include symmetrical diethylhydrazine, SDEH, N,N'-diethylhydrazine and hydrazoethane (IARC, 1974). 1,2-Diethylhydrazine will be referred to as 1,2-DEH throughout this profile. The structure of 1,2-DEH is given below:

$$CH_3-CH_2-NH-NH-CH_2-CH_3$$

Molecular formula: $C_4H_{12}N_2$

Molecular weight: 88.2

The Chemical Abstract Service (CAS) Registry number for 1,2-DEH is 1615-80-1.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

1,2-DEH is a colorless liquid at ambient temperature. Some of the physical and chemical properties of 1,2-DEH are given below (IARC, 1974):

Boiling point:	85-86°C
Density (at 20°C):	0.797
Solubility:	Unlike the dimethyl isomer, this compound is not soluble in water but soluble in ethanol, diethyl ester and benzene (Weast, 1980). The hydrochloride is soluble in water.
Stability:	The free base is highly reactive and oxidizes readily.

1.3. PRODUCTION DATA

1,2-DEH is not produced commercially other than as a research chemical (IARC, 1974). Specific production data were not located in the available literature (see Appendix). 1,2-DEH may be produced during the thermic decomposition of ethylamine (Taylor and Ditman, 1936).

1.4. USE DATA

There are no known commercial uses for 1,2-DEH, although it has been evaluated experimentally as a high-energy rocket propellant (Stauffer, 1977). The compound is used in chemical laboratories, such as for the synthesis of symmetrical di-Mannich bases (IARC, 1974).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

Information on the environmental fate and transport of 1,2-DEH are not available, presumably due to the limited (i.e., non-commercial) production and use. Structure-reactivity principles (Mill, 1979) can be used, however, to predict the behavior of 1,2-DEH in the environment from information that is available for lower hydrazines. Data for hydrazine, methylhydrazine and 1,1-dimethylhydrazine indicate that reaction with ozone and hydroxyl radicals may be the major fate of 1,2-DEH in the atmosphere (Tuazon et al., 1981, 1982; Pitts et al., 1981). Based on kinetic data for these hydrazines, it is expected that the half-lives for reaction of 1,2-DEH with ozone and hydroxyl radical will be on the order of several minutes and several hours, respectively. Photolysis is not expected to occur in the solar actinic region (λ >290 nm) (Tuazon et al., 1981). In water, oxidation of 1,2-DEH by dissolved oxygen is anticipated to be a major pathway for chemical degradation; data for methylhydrazine and 1,1-dimethyl-hydrazine suggest a half-life on the order of 1 day (Banerjee et al., 1977, 1981). Upon release to soil, 1,2-DEH is expected to be rapidly oxidized by gaseous or dissolved oxygen. Leaching of this compound from soil may be less likely because it is not soluble in water.

3. EXPOSURE

Monitoring data for 1,2-DEH in the air, water or food were not found in STORET or the available literature (see Appendix). The presumed high reactivity of 1,2-DEH suggests that this compound will be rapidly degraded in the environment via chemical pathways and that ambient exposure will be negligible.

4. PHARMACOKINETICS

4.1. ABSORPTION

Pertinent data regarding the absorption of 1,2-DEH were not located in the available literature (see Appendix).

4.2. DISTRIBUTION

Pozharisski et al. (1975) found that peak concentrations of radioactivity in the blood of rats occurred 3 hours after the subcutaneous (s.c.) injection of 21 mg/kg of 1,2-di(^{3}H-ethyl)hydrazine. The level in blood subsequently decreased to <0.50 the 3-hour value by 6 hours post-injection. Marked radioactivity was found in the blood, bile and urine following treatment, but radioactivity was not detected in any part of the gastrointestinal tract (i.e., stomach, duodenum, ileum, descending and ascending colon). A similar pattern of blood distribution reportedly occurred following oral administration of the same dose of labeled 1,2-DEH. Further studies with rats showed that 1,2-di(^{3}H-ethyl)hydrazine did not alkylate macromolecules in the intestinal mucosa, liver or kidneys 3 or 9 hours after s.c. injection, although radioactivity at the same times in the thymus, spleen and brain (which are target organs for carcinogenesis) was high (Pozharisski et al., 1975).

4.3. METABOLISM

Druckrey (1968a, 1974) has postulated that 1,2-DEH is an indirect carcinogen that is activated in the liver via formation of azoethane, azoxyethane, ethylazoxyethanol and ethyldiazonium ion; ethyldiazonium ion, a highly reactive species, forms ethylcarbonium ion which presumably is the ultimate alkylating carcinogen. This metabolic scheme is inferred from the positive results of carcinogenicity studies of the postulated intermediates, which showed organo-specific patterns of carcinogenesis similar to 1,2-DEH.

Activation in the liver is indicated by the specific induction of liver tumors following oral administration of 1,2-DEH (Druckrey, 1968a); s.c. or intravenous (i.v.) injection produced malignant tumors in the brain, olfactory bulbs, mammary glands and hematopoietic system, but not in the liver.

4.4. EXCRETION

Radioactivity was detected in the urine and bile of rats following the s.c. injection of 21 mg/kg 1,2-di(^{3}H-ethyl)hydrazine (Pozharisski et al., 1975). Peak concentrations occurred 6 hours post-injection, which was the maximum sampling time after administration.

5. EFFECTS

5.1. CARCINOGENICITY

Weekly s.c. injections of 25, 50 or 100 mg/kg of 1,2-DEH dihydrochloride given to BD rats for 30 weeks produced tumors that were localized in the brain, olfactory bulbs, mammary glands and hematopoietic system (Druckrey et al., 1966). When administered orally to rats, 1,2-DEH reportedly produced hemangioendotheliomas of the liver with high specificity, and multiple metastases in the lungs (Druckrey, 1968a). Additional data regarding these experiments were not provided.

When single doses of 50 or 150 mg/kg 1,2-DEH were injected intravenously into female BD rats on day 15 of gestation, tumors of the brain, spinal cord or peripheral nerves developed in 18/19 and 11/12 offspring, respectively (Druckrey et al., 1968). The transplacental carcinogenicity of 1,2-DEH was confirmed in a subsequent study with a larger number of animals (Druckrey, 1973a); single i.v. doses of 10 and 50+ mg/kg on day 15 of pregnancy elicited development of the same types of malignant nervous system tumors in 7/20 and 39/42 surviving offspring, respectively.

The design and results of the above studies are summarized in Tables 5-1 and 5-2, but control data were not reported in any of the aforementioned studies.

Postulated metabolites of 1,2-DEH, azoethane and azoxyethane, have been shown to be carcinogenic to the same species of rats following injection and/or inhalation (Druckrey 1968a,b, 1973b; Druckrey et al., 1965, 1967; Preussman et al., 1969).

5.2. MUTAGENICITY

Pertinent data regarding the mutagenicity of 1,2-DEH were not located in the available literature (see Appendix).

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TABLE 5-1

Summary of Subcutaneous Carcinogenicity Study of 1,2-Diethylhydrazine in BD Rats[a,b]

Dose or Exposure	Duration of Treatment	Duration of STudy	Purity of Compound	Vehicle or Physical State	Target Organ	Tumor Type	Tumor Incidence (p value)
25, 50 or 100 mg/kg[c] once weekly	30 weeks	NR[d]	NR	neutralized aqueous solution	NA	tumors	43/45
					brain	gliomas	6/45
					olfactory bulbs	neuroaesthesio-epitheliomas or blastomas	15/45
					mammary gland	adenocarcinoma	15/20
					hematopoietic system	leukemias or malignant reticulocytes	13/45

[a]Source: Druckrey et al., 1966

[b]Sex of rats not reported; no control data were reported, and tumor incidences were not tabulated by dose level.

[c]Administered as 1,2-diethylhydrazine dihydrochloride and calculated as the base. The total doses were reported to be 770, 1400 and 2700 mg/kg.

[d]The average tumor latency periods ranged from 250 days (low dose) to 215 days (high dose).

NR = Not Reported; NA = Not Applicable.

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TABLE 5-2

Summary of Intravenous Carcinogenicity Study of 1,2-Diethylhydrazine in Offpsring of Female BD Rats[a,b]

Dose or Exposure (mg/kg)	Duration of Study	Purity of Compound	Vehicle or Physical State	Tumor Type	Tumor Incidence (p value)
150	gestation and lifetime of progeny	NR	neutralized aqueous solution	malignomas of the brain, spinal cord or peripheral nerves[c]	11/12[d] offspring
50	gestation and lifetime of progeny	NR	neutralized aqueous solution	malignomas of brain, spinal cord or peripheral nerves[e]	18/19[d] offspring
50+	NR	NR	NR	malignomas[f] offspring	39/42
10	NR	NR	NR	malignomas[f] offspring	7/20

[a]Sources: Druckrey et al., 1968; Druckrey, 1973a

[b]The target area in each case was the nervous system and the duration of treatment was a single dose on day 15 of gestation.

[c]A total of 11 brain, 2 spinal cord and 5 peripheral nerve tumors was observed.

[d]11/12 and 18/19 of the offspring died between days 126 and 498 with tumors of the brain, spinal cord or nervous system.

[e]A total of 4 brain, 2 spinal cord and 12 peripheral nerve tumors was observed.

[f]Most of the tumors occurred in the brain (gliomas and ependymomas) and peripheral and cranial nerves (neurinomas); some occurred in the spinal cord.

NR = Not Reported

No control data were reported, and the route of exposure was not natural. The progeny were observed for their lifespan.

5.3. TERATOGENICITY

Druckrey (1973a) found that 1,2-DEH is a potent teratogen when administered to pregnant BDIX rats by single i.v. injection. Details of the experimental protocol and results were not reported, but administration on the 10th day of gestation partially or completely inhibited development of the eyes and optic nerves (often resulting in congenital blindness) at "low" doses, and induced hydrocephalus in the range of "medium" doses. Microphthalmia and anophthalmia apparently never occurred spontaneously in BDIX rats. When administered between the 12th and 15th day of gestation at a "high" dose, 1,2-DEH induced malformations of the paws, but cleft palates were not observed. The incompletely reported results are part of systematic studies on organo-specific carcinogenesis and teratogenesis that involved a number of indirect alkylating methyl and ethyl compounds; the teratogenicity studies were mainly directed to induction of malformations of the brain and sensory organs.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of 1,2-DEH were not located in the available literature (see Appendix).

5.5. CHRONIC TOXICITY

Pertinent data regarding the chronic toxicity of 1,2-DEH were not located in the available literature (see Appendix).

5.6. OTHER RELEVANT INFORMATION

Pertinent data regarding other relevant information were not located in the available literature (see Appendix).

6. AQUATIC TOXICITY

Pertinent data regarding the toxic effects of 1,2-DEH to aquatic organisms were not located in the available literature (see Appendix).

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Recommended or promulgated occupational or environmental exposure limits for 1,2-DEH were not located in the available literature (see Appendix).

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of 1,2-diethylhydrazine were not located in the available literature (see Appendix).

8. RISK ASSESSMENT

1,2-DEH has been shown in studies lacking control groups to be carcinogenic to rats by s.c. injection (weekly) and by transplacental exposure (single intravenous injections to dams during pregnancy). The tumors were localized in the nervous system (s.c. and transplacental exposure) and in the mammary glands and hematopoietic system (s.c. exposures). Oral administration of 1,2-DEH has been reported to specifically induce hemangioendotheliomas in the liver of rats. Further, postulated metabolites of 1,2-DEH show similar patterns of organo-specific carcinogenesis.

The available evidence is sufficient to implicate 1,2-DEH as an animal carcinogen. The available studies cannot be used for derivation of a q_1^*, however, because incidence data and details of the experimental design were not reported (i.e., the oral study), irrelevant routes of exposure were employed, and control data were not reported (i.e., the s.c. and transplacental studies).

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APPENDIX: LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
EPCASR
Chemical Industry Notes

Most of these searches were conducted in February, 1983; a few were conducted March-May, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 7 and 8th), and the following secondary sources were reviewed:

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